**Intro into Pharmacology Objectives**

1. Define “Pharmacology”, “pharmacodynamics” and “pharmacokinetics”.
   * **Pharmacology**—the study of substances that interact with living systems through chemical processes, especially by binding to regulatory molecules and activating or inhibiting normal body processes
   * **Pharmacodynamics**—The study of the drug effects on the body
     + What does it do?—dose response relationship
     + How does it work? –interactions of drugs with receptors
   * **Pharmacokinetics**—the study of how the body affects the drug and its travels through the organism
     + Time-action relationship
     + Drug absorption, distribution, biotransformation, elimnination
2. Describe, using both words and graphics, the interactions between drugs and their receptors, including such factors as affinity, efficacy, and signal transduction.
   * Drug receptors—macromolecules at which a drug first interacts to eventually affect cell function
     + A drug may interact with more than one type of receptor
     + many drugs have chirality but only one stereoisomer has significant biological activity
   * The probability that a drug receptor interaction will occur is a function of drug concentration…..
     + affinity
       - Affinity is the measure of binding strength between the drug molecule and its binding site on the receptor
       - Drug molecules are constantly binding (associating with and unbinding (dissociating) from receptors
       - The effect of the agonist on the cell depends on the affinity of the receptor for the drug and the drug concentration
       - When drugs bind with different affinity to the same receptor they differ in their potency
         * Potency is seen by where the curve (log(conc. Of the agonist) by the E/Emax) is on the X axis (log of the concentration of the agonist)
       - Is independent of efficacy
     + Efficacy
       - Efficacy is the ability to cause the receptor to change to the active state
       - Values range from 0🡪1
       - Efficacy is reflected by the maximum effect produced so it affects the Y axis (E/Emax) of the curve (log(conc. Of the agonist) by the E/Emax). Which would be the ceiling of the curve
       - Is independent of affinity
     + Signal transduction
       - Amplification of the signal
       - Ex: cAMP as a second messenger
3. Describe, using both words and graphics, the factors that describe the relationship between drug concentration (and drug dose) and the response.
   * The number of receptors bound at any moment is a function of the concentration of drug and the affinity of the receptor for the drug
   * Equilibrium dissociation constant (KD)—the concentration of drug at which half the receptors are bound when the system has reached equilibrium
   * In most cases there is amplicfication during the transduction process, so that the effect is relatively rgreat than the fraction of receptors occupied and the maximum response can be obtained with <100% of the receptors occupied.
4. Describe, using both words and graphics, the interactions between agonists, partial agonists, and competitive and non-competitive antagonists.
   * Agonist—affinity and full efficacy (=1)
     + The effect of the agonist on the cell depends on the affinity of the receptor for the drug and the drug concentration
     + EC50= the agonist concentration when E/Emax is 0.5 (half the maximal response)
   * Partial agonist—affinity and efficacy >0, but <1
     + Acts on the same receptor system as the full agonist but has a lower maximal efficiacy regardless of dose
     + Partial agonist may be more potent, less potent, or equally potent (b/c potency is an independent factor)
   * Competitive antagonist—affinity but efficacy =0. Interacts in a reversible fashion with the same recognition site on the receptor as the agonist. The interactions are determined by the concentration and affinities of the drugs
     + In a log conc-effect curve adding a competitive agonist decreases potency (so shifts the curves to the R) but no change in efficacy
     + Ex: atropine is a competitive antagonist t muscarinic cholinergic receptors
   * Non-competitive antagonist
     + May bind irreversibly to the same recognition site as the agonist OR may act at a different site on the receptor molecule (allosteric interaction)
     + This has no effect on potency but depressed the maximum agonist effect. Decreases efficacy.
5. Compare the uses of graded and quantal dose-response curves, and be able to compare drugs based on the ED50, LD50, and related terms
   * The log dose-response curve describes the relationship between the dose administered and the response
   * 2 ways to measure drug effect—
     + graded effect
       - quite similar to the log-conc effect curve
       - describes the size of the effect produced by various doses of drug (ex: how much did the BP decrease after 10, 20, 30, or 40 mg of propranolol?)
       - will see at what does you see a certain intensity of a certain effect. Can plot multiple drugs together.
       - Can also plot multiple effects of a single drug
       - Answers
         * How much effect did this dose of drug cause in the patient
         * What is the average size of the effect of this dose?
     + quantal effect
       - derived from data that answer the question
         * “did this dose of the drug have an effect in the patient?”,
         * in a population, “what is the probability that this dose will produce the effect in someone?” For example, “What % of our patients had a decreased blood pressure of at least 10 mm Hg after 10, 20, 30 or 40 mg of propranolol?”
         * y axis does NOT show size of the drug effect but does show fraction or % of population responsing
         * ED50—the dose at which half the population responds to the drug
         * TD50—the median toxic dose
         * LD50—median lethal dose
   * Therapeutic index—TD50/ED50
     + This single ratio is usually not enough to judge the safety of a drug
     + Safer drugs have higher TI values